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Measurement of Field Resistance, Rust Hazard, and Deployment of Blister Rust-Resistant Western White Pine

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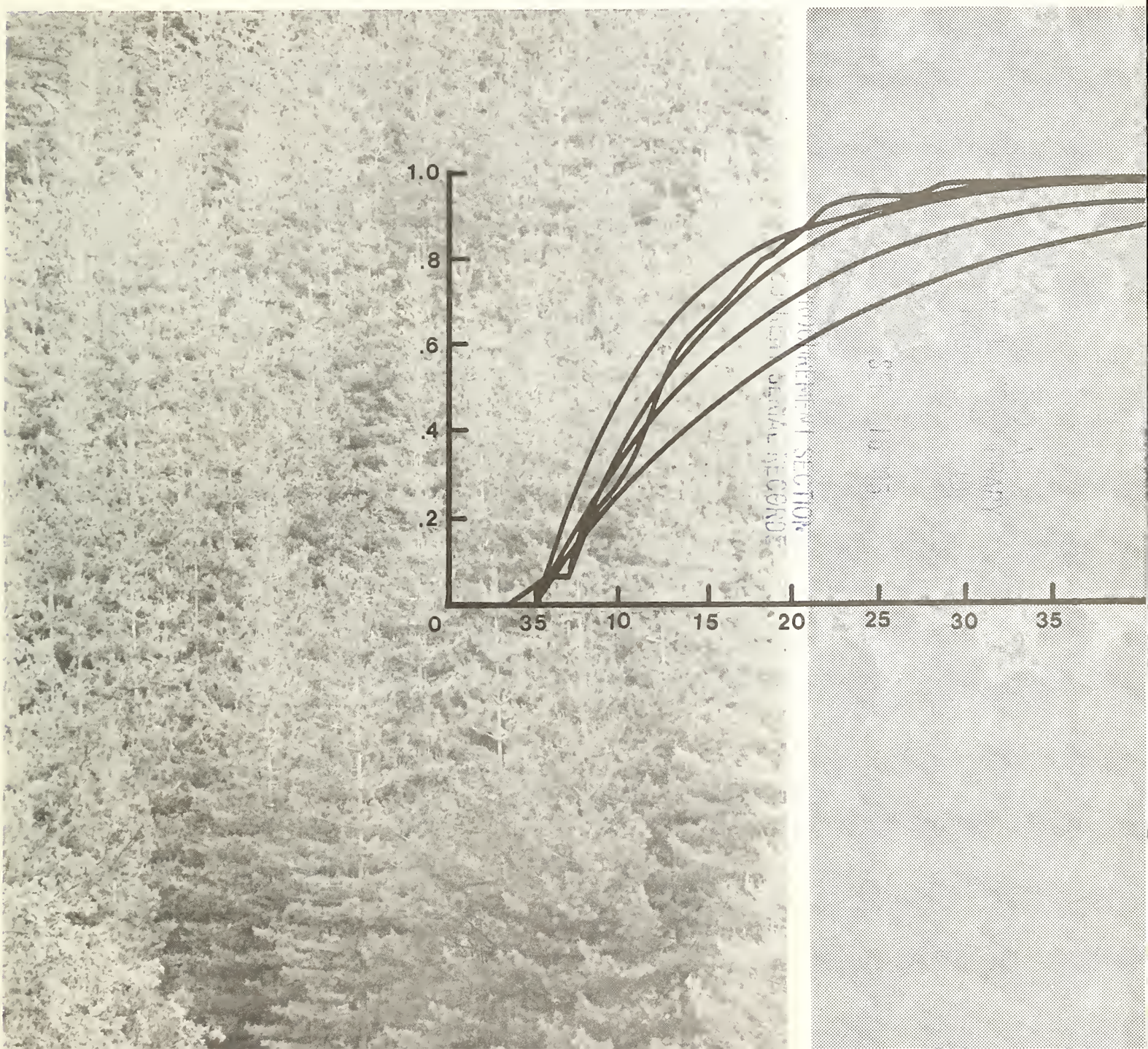

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RESEARCH SUMMARY

Three white pine plantations, composed of materials of several levels of resistance from early generations of the blister rust resistance breeding program, were compared on the basis of annual infection and mortality rates. The sites varied in blister rust hazard; resistant materials were consistent in their performance, and the infection data behaved as one would expect if the genetic and environmental assumptions of the simple interest disease progress model were violated.

Presumably, the resistant populations violate the genetic assumptions to the extent of their resistance, and any assessment of resistance performance must take into account both environmental as well as genetic violation. Field performance is discussed in relation to measuring hazard on wild seedlings.

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INTRODUCTION

Soon after the white pine blister rust fungus (*Cronartium ribicola* J.C. Fisch. ex Rabenh.) was discovered on western white pine (*Pinus monticola* Dougl.) in the Inland Empire, plant quarantines were established in an attempt to prevent further introductions. In spite of efforts to control the intensification and spread of the disease by eliminating the obligate alternate host, *Ribes* species, the epidemic continued until virtually all the area had been exposed. The *Ribes* eradication programs were continued in the hope that losses to the disease could be held to an acceptable level. At the same time, hopes were that numerous studies on various aspects of the ecology, physiology, and pathology of the disease would lead to new control measures. Attempts at direct chemical control of the disease in infected trees were one result of that research. However, in 1967, after nearly four decades of control efforts, researchers concluded that none of the control measures were sufficiently effective to warrant their continuation (Ketcham and others 1968).

Although efforts to control the rust fungus were not successful, efforts to produce trees resistant to the rust are showing considerable promise (Bingham and others 1960). Since 1970, approximately 1,300 lb of seed containing low to moderate levels of rust resistance have been produced in a research breeding arboretum and a grafted seed orchard. Additional orchards designed to produce seeds containing higher levels of resistance are beginning to produce (Hoff and McDonald 1980), but the demand still exceeds the supply. Even if there were enough high-quality seed, it might not be good policy to challenge the rust with only highly resistant trees because this could result in widespread selection for more virulent fungal races. Indeed, at least one new race has already made an appearance (McDonald and others 1984).

A single response that would address both shortage of resistant seed and maintenance of genetic breadth is alignment of resistance to hazard (McDonald and Hoff 1982). This response, of course, presumes that hazard does indeed vary. A tour through northern Idaho western white pine stands readily demonstrates that 60 years after introduction of *C. ribicola*, the epidemic is not of equal intensity and impact. Of the many elements of the disease complex, the most important seems to be the number, distribution, and species of *Ribes* (McDonald and others 1981). We know ribes populations vary greatly from thousands of bushes per acre to total absence. Records from the early ribes eradication program (USDA 1950) indicated more ribes-free stands of white pine as well as lower densities (table 1) in the Kaniksu National Forest where 46 percent of the white pine stands were ribes free, in contrast to the more southerly St. Joe National Forest with only 15 percent ribes-free stands. (The two forests today are part of the Idaho Panhandle National Forests.)

There is also evidence of variation in hazard within a given drainage. For example, on the Priest River Experimental Forest replicate plantings on three sites were established in 1971 to investigate genetic variation within and among stands of white pine (Steinhoff 1979). Blister rust infection (percentage of stems infected),

Table 1.—Ribes eradicated per acre with the first working, 1923 to 1950

Operation	Gross acres	Mean number of ribes plants per acre					
		R. lacustre	*R. viscosissimum*	*R. petiolare*	*R. inermi*	Others	Total
Clearwater	423,980	95	123	8	2	*	228
St. Joe	542,520	86	152	6	6	*	244
Coeur d'Alene	347,030	111	46	*	14	1	172
Kaniksu	492,600	47	65		9	*	121

*Less than 1 per acre.

mortality, and ribes abundance on these sites indicate (McDonald and Hoff 1982) that hazard varies a great deal over distances of less than 1 mile, and that relative abundance and species of local ribes plants is closely associated with degree of damage.

This paper reports on field performance of western white pine trees with various levels of heritable resistance to blister rust in plantations where the rust hazard ranged from moderate to high. Field resistance was estimated two ways: reduced infection rate (percentage infected per year) and lower proportion dead.

MATERIALS

The blister rust resistance breeding program was started about 1950. Western white pine trees selected for phenotypic resistance were mated, and the resulting progenies were inoculated in nursery beds to determine genotypic resistance (Bingham and others 1969). Open-pollinated cones were also collected from the selected trees and the resulting seedlings used in the resistance testing program.

Excess seedlings from controlled and open pollinations were used to establish field plantings at Priest River Experimental Forest, Deception Creek Experimental Forest, and at Emerald Creek near Clarkia, ID. Planting was done in 1955, 1956, 1957, and 1959 with 22 to 45 lots established per year in three blocks at each location. The number of seedlings available varied greatly from two to 24 per lot per location. Open-pollinated control lots from cankered and presumably nonresistant trees growing with the phenotypically resistant selections were included in each planting.

Because of the unbalanced design, critical analysis of individual lots is of doubtful value. However, the individual parents were classified on the basis of nursery inoculations (Bingham and others 1969), and their progenies were grouped into five resistance classes as follows:

1. Crosses between good general combiners (GCA × GCA).
2. Crosses involving at least one parent not classed as a good combiner (Other Crosses). Most of these involved one GCA parent.
3. Open-pollinated progeny of GCA's (GCA OP).
4. Open-pollinated progeny of parents not classed as good combiners (Other OP).
5. Susceptible controls (Controls).

Records from previous examinations were surveyed, and any trees recorded as severely infected or rust-killed at an earlier date but not present in 1980 were added to the rust mortality count. Trees killed directly or indirectly by blister rust were included in counts of infected trees.

Naturally regenerated white pines were found in fair abundance within the plantings at each location. These volunteers ranged in age from less than 10 years to about 23 years. The rust condition (including rust-killed trees) of a sample of volunteer white pines was recorded at each location. For each wild tree sampled, the age was estimated by count of annual whorls.

Field performance was assessed as proportion infected and proportion dead in 1980. Plant disease epidemics usually develop (see Pfender 1982) according to either a simple or compound interest model (Van der Plank 1975; Zadoks and Schein 1979). Knowledge about the biology of western white pine blister rust leads to the conclusion that this system fits the simple interest model. This model yields disease progress curves that eventually reach 100 percent infection if the following assumptions are met (Baker and Drury 1981): (1) a random distribution of inoculum, (2) a random occurrence of infection conditions, and (3) all members of the host population are susceptible to the pathogens. Many times epidemics do not meet these assumptions (Grogan and others 1980).

Using McDonald and Hoff's simple interest formula for eventual 100 percent infection:

$$r = \frac{1}{t_2} \log e \frac{1}{1 - X_2} \quad (1)$$

where

t_2 = time of measurement

X_2 = proportion infected,

the infection rates through 1970 for each resistance class in the Priest River and Deception Creek plantings were calculated from data reported by Steinhoff (1971). These rates were used to project expected proportion of trees infected in 1980:

$$X = 1 - e^{-rt_3} \quad (2)$$

where

X = expected proportion infected at time t_3 = (1980)

r = infection rate calculated from equation 1.

The calculated expected infections and observed 1980 infections were used in chi-square analysis to assess the fit of the simple interest model.

Detection of incipient white pine blister rust cankers during the first year after infection is impossible because many cankers require more than 1 year to appear. Because older cankers are easily seen, 2 years were arbitrarily deducted from the time since planting to estimate "years of exposure" for calculation of annual infection rates. Infection rates calculated on the basis of 1980 data for all resistance classes, establishment years, and locations were subjected to analysis of variance.

Mortality percentages for the various classes, years, and locations, transformed to arcsin of the square root, were similarly analyzed.

RESULTS

This study yielded three principal results: (1) expectations relative to the simple interest model of disease progress, (2) temporal and spatial variation of infection rates, and (3) spatial and temporal variation of mortality levels.

The Simple Interest Model for Disease Progress

Chi-squares from the observed and expected infections based on the simple interest formula are listed in table 2. Considering all progeny categories, establishment years, and locations, the total and pooled chi-squares are high and indicate highly significant departure of the observed

Table 2.—Chi-square analysis of total observed blister rust infection in white pine resistance classes at two locations and infection expected on the basis of 1970 infection rates projected by the simple interest model

Location and infection year	Resistance class	Number of trees	1970 infection rate	1980 infection		Chi-square
				Expected No.	Observed No.	
PR-1955	GCA × GCA	137	0.0169	44.1	39	0.59
	Other Crosses	111	.0185	38.5	31	1.46
	GCA OP	96	.0277	45.2	39	.85
	Other OP	48	.0443	30.7	34	.35
	Controls	23	.0640	17.7	17	.03
PR-1957	GCA × GCA	63	.0281	28.1	19	2.95
	Other Crosses	108	.0339	67.2	52	3.44
	GCA OP	126	.0436	75.6	70	.41
	Other OP	39	.0868	32.7	28	.68
	Controls	60	.0761	47.9	41	.99
PR-1959	GCA × GCA	91	.0288	38.3	31	1.39
	Other Crosses	122	.0365	61.0	60	.02
	GCA OP	39	.0522	30.6	19	4.40
	Other OP	70	.0837	55.7	51	.40
	Controls	31	.0603	21.1	20	.06
DC-1955	GCA × GCA	154	.0192	55.0	48	.89
	Other Crosses	138	.0346	75.8	62	2.51
	GCA OP	95	.0591	70.6	64	.62
	Other OP	43	.0761	35.5	34	.06
	Controls	34	.1336	32.4	32	.01
DC-1957	GCA × GCA	86	.0385	47.6	34	3.89
	Other Crosses	159	.0394	89.5	61	9.08
	GCA OP	174	.0477	110.0	88	4.40
	Other OP	38	.0909	32.4	29	.36
	Controls	88	.1074	78.7	75	.17
DC-1959	GCA × GCA	86	.0363	42.9	28	5.18
	Other Crosses	111	.0320	50.5	38	3.09
	GCA OP	37	.0480	22.2	16	1.73
	Other OP	70	.1018	59.5	47	2.78
	Controls	27	.0813	21.2	17	.83

	All classes			Susceptible classes only (Other OP and Controls)			
	df		Prob.		df		Prob.
$\Sigma\chi^2$	30	53.60	0.005	$\Sigma\chi^2$	12	6.72	0.85
Pooled χ^2	1	37.73	.001	Pooled χ^2	1	3.59	.05
Heterogeneity χ^2	29	15.87	.98	Heterogeneity χ^2	11	3.13	.98

from the expected infection frequencies (table 2). Almost all of the observed frequencies are lower than the expected. Because the heterogeneity chi-square has a high probability of being larger, the consistency of overestimation is confirmed.

Note, however, that the two most susceptible classes, Other OP and Controls, generally contribute relatively little to the total chi-square. Considering these two classes alone, the chi-square of 6.72 is a value that could be obtained by chance over 85 percent of the time, meaning that estimate and actual are not significantly different. The heterogeneity chi-square value for this subgroup (probability of a larger χ^2 by chance is 0.98) still indicates consistent overestimation.

Variation of Infection Rates

Although average annual infection rates as calculated did not provide highly reliable predictions of future infections, except for nonresistant trees, they did standardize infection data by removing effects of varying periods of exposure to the disease and provided a more stable measure of disease intensity up to the time of assessment. Infection rates based on data through 1980 (table 3) varied significantly among resistance classes and locations (table 4). The effect of planting year was not significant. Mean infection rates at Emerald Creek were significantly higher than those at Priest River at the 5 percent level (Duncan's multiple range test), and rates at Deception Creek were intermediate (table 3).

Table 3.—Total infection and infection rate by resistance class and planting location

Resistance classes	Priest River		Deception Creek		Emerald Creek		Mean[1]	
	Total infection	Infection rate	Total infection	Infection rate	Total infection	Infection rate	Total infection	Infection rate
	Percent		*Percent*		*Percent*		*Percent*	
GCA × GCA	30.9	0.018	34.4	0.019	46.7	0.031	37.3	0.026ab
Other Crosses	38.5	.024	39.2	.024	46.8	.030	41.2	.026ab
GCA OP	48.3	.032	53.7	.037	61.7	.049	54.6	.040b
Other OP	71.8	.061	74.2	.065	73.4	.071	73.1	.066c
Controls	68.9	.056	80.8	.089	86.0	.095	78.6	.080c
Mean[1]	51.7	.038a	56.5	.047ab	62.9	.055b		

[1]Column and row means of infection rates with different letter superscripts are significantly different at the 5 percent level of probability.

Table 4.—Analysis of variance of mean annual rate of blister rust infection through 1980 in progenies established at three locations in three planting years

Source of variation	Degrees of freedom	Sum of squares	Mean square	F value
Total	44	0.0337		
Years (Y)	2	.0001	0.00007	NS
Locations (L)	2	.0021	.00107	3.82*[1]
Resistance classes (RC)	4	.0226	.00565	20.2**
Residual	36	.0099	.00028	

[1]* and ** indicate significance at the 5 and 1 percent levels, respectively.

Differences among resistance classes were consistent with expectation; over two-thirds of the total sum of squares in infection rates was due to resistance classes with the following contrasts (Duncan's multiple range test), significantly contributing to variation (table 3):

1. Infection rate of all select progenies combined was lower than that of the controls.
2. Progeny of controlled crosses had lower rates than open-pollinated progenies.

Note that the mean rate for open-pollinated progenies of trees other than the GCA's was not significantly lower (Duncan's multiple range test) than the controls and far higher than the rate for controlled crosses among these trees.

Rust data from white pine natural regeneration within the plantings were not included in the analysis of variance and, therefore, cannot be statistically compared with the planted trees. In most instances, however, the infection rate for volunteers was higher than that of planted trees, including the nonresistant control lots (table 5). At Deception Creek, the infection rate of young volunteer age classes was relatively low in contrast to other nonresistant classes.

Table 5.—Infection rate and mortality of naturally seeded volunteer white pines at three locations in contrast to planted susceptible controls

	Planted controls	Volunteers 11-20 yr exposure
Priest River		
Annual infection rate	0.056	0.089
Mortality (percent)	20.1	14.9
Number of trees	114	76
Deception Creek		
Annual infection rate	0.089	0.062
Mortality (percent)	27.1	16.7
Number of trees	149	1,221
Emerald Creek		
Annual infection rate	0.095	0.353
Mortality (percent)	27.9	11.1
Number of trees	43	48

Deception Creek - volunteers by age class

Number of trees	Years exposure	Infection rate	Mortality (percent)
121	6-10	0.042	8.3
630	11-15	.047	11.2
591	16-20	.078	22.2
14	21-25	.089	35.2

Table 6.—Percentage rust-related mortality by resistance class, location, and planting year

Resistance class	Planting location	Planting year 1955	1957	1959	Mean[1]
GCA × GCA	Priest	3.6	12.7	13.2	9.8
Other Crosses	River	4.5	6.5	10.7	7.2
GCA OP		4.2	11.1	17.9	11.1
Other OP		10.4	10.3	22.9	14.5
Controls		26.1	8.3	25.8	20.1
Mean		9.8	9.8	18.1	12.5[x]
GCA × GCA	Deception	11.7	9.3	14.0	11.7
Other Crosses	Creek	15.9	16.4	13.5	15.3
GCA OP		20.0	17.8	21.6	19.8
Other OP		20.9	34.2	35.7	30.3
Controls		17.6	34.1	39.6	27.1
Mean		17.2	22.4	22.9	20.8[y]
GCA × GCA	Emerald	4.5	20.0	27.1	17.2
Other Crosses	Creek	10.2	7.1	22.9	13.4
GCA OP		0	17.9	41.2	19.7
Other OP		7.7	10.0	25.7	14.5
Controls		14.3	10.5	58.8	27.9
Mean		7.4	13.1	35.1	18.5[y]
GCA × GCA	All	6.6	14.0	18.1	12.9[a]
Other Crosses		10.2	10.0	15.7	12.0[a]
GCA OP		8.1	15.6	26.9	16.9[a]
Other OP		13.0	18.2	28.1	19.8[ab]
Controls		19.3	17.6	38.1	25.0[b]
Mean[1]		11.4[a]	15.1[a]	25.4[b]	

[1]Resistance class, location, and year means with different letter superscripts are significantly different at the 5 percent level of probability.

Table 7.—Analysis of variance of rust-caused mortality through 1980

Source	Degrees of freedom	Sum of squares	Mean square	F
Total	44	3,419		
Locations (L)	2	344	172	3.25**[1]
Years (Y)	2	966	498	9.40*
Resistance classes (RC)	4	563	141	2.66**
Residual	36	1,915	53	

[1]* and ** indicate significance at the 5 and 1 percent levels, respectively.

Variation of Mortality Percentage

The percentage of trees killed by *C. ribicola* since establishment of the plantings was recorded by resistance class, location, and year (table 6). As was the case for infection rate, clear, consistent differences between resistance classes are evident. Analysis of variance (table 7) indicated that all major effects (resistance class, location, and establishment year) were significant. As was the case in infection rates, rust mortality was much lower at Priest River than at the other two locations (table 6).

In contrast to infection rates, the strongest single cause of variation in mortality was planting year. Rust mortality of trees planted in 1959 was approximately double that of trees planted in 1955 and 1957. Mortality was also unexpectedly high in the 1957 planting at Deception Creek (table 6) and indicates that significant year × location interaction could occur.

DISCUSSION

The results of this study indicate that the simple interest formula provides a reasonably accurate prediction of future infection of susceptible classes even though, for these three sites, there was a consistent overestimate. The simple interest model would overestimate: if spore distribution was nonrandom (Fracker 1936; Grogan and others 1980; Baker and Drury 1981); if environment at infection was nonrandom (Baker and Drury 1981); or if host or rust genotypes were nonrandom in distribution (Grogan and others 1980; Baker and Drury 1981). The consistently large overestimate of infection of resistant progenies also fits the above explanations. Presumably, there are some susceptible individuals among the progenies of resistant parents. These susceptible individuals become infected fairly early in life, whereas their resistant siblings remain free of rust even after a number of years of exposure. As a result, the family as a whole does not follow the expected ever-upward infection curve as predicted by the interest formula. In short, the potential infection in the resistant classes appears to be much below 100 percent. Our results are consistent with those reported for black rot of peanut and fusiform rust of southern pines (Hanounik and others 1977; Griggs and others 1978).

Whether judged on the basis of infection percentage or rate of infection, mating of trees with high general combining ability for resistance produced progeny with proportionally fewer susceptible individuals than other classes, and all classes performed relatively consistently over the various planting sites and years.

Variation also occurred in the mortality among resistance classes. Most of this variation can be attributed to high mortality of control lots in comparison to all other classes. Also, control crosses generally had lower mortality than open-pollinated lots. However, the greatest single factor affecting variation in mortality was planting year.

Resistant families should deviate from the expected just as families resistant to fusiform rust deviated (Griggs and others 1978) and families of sugar pine resistant to blister rust deviated (Kinloch 1981). On the other hand, we should expect some sites to give the expected 100 percent infection of susceptible material (Kinloch 1981).

Rust-related mortality of trees of GCA ×, GCA, and GCA OP resistance classes planted in 1957 at Emerald Creek and at all but the GCA × GCA class at Deception Creek in 1957 and in 1959 was much higher than that of trees planted at Priest River. In the case of trees planted in 1959 at Deception Creek and Emerald Creek, mortality was consistently higher than it was at Priest River for all classes. These data follow the patterns expected if a year favorable for rust infection occurring soon after stand establishment results in much higher mortality than severe rust years that occur later in the life of the stand (McDonald 1979), even though average annual infection rates are comparable.

Mortality necessarily is related to infection because a tree must be infected before it can be killed by the disease. However, in the current study many (in fact, most) infected trees did not die. There was little difference in proportion of trees infected by planting years with total infection ranging from 48 to 53 percent. In contrast, rust-caused mortality varies greatly, depending on year of establishment, resistant class, and location. Consequently, mortality was not closely related to infection rate. The correlation coefficient of mortality with infection rate was a significant 0.51, but this explains only about a fourth of the variation in mortality.

Further correlation analysis suggests that the more resistant classes are less subject to the year-to-year shift in mortality rate than are the highly susceptible classes. Correlation coefficients were 0.24 for the Controls and Other OP classes and 0.67 for the more resistant classes. An important conclusion is that susceptible trees are much more likely to be killed by heavy early infection than are resistant trees. Resistance decreases both infection rate and early mortality.

The mean infection rates and total infection percentages in 1980, 21 to 25 years after planting, clearly indicate that blister rust was more severe at Emerald Creek than at the other two locations (table 3). Mortality data also indicate the lesser hazard at the Priest River planting (table 6). But although these results today demonstrate the differences in rust hazard at these locations, how could a forest manager in 1955 have determined the relative hazard? And how could a manager today determine the hazard at other planting sites?

Disease incidence data from the volunteer trees should provide an independent assessment of the relative hazard at the three locations. However, our preliminary results (table 5) are more indicative of sampling problems than of the comparative hazard.

The small sample of volunteer pines taken at Emerald Creek provided only two to 10 trees per 1-year age class. Although these numbers appear to be inadequate, as most trees of all age classes were infected, perhaps a reasonable depiction of high hazard was presented. The mortality rate seems low, but volunteers were younger than planted trees. Time of exposure and age × infection year interaction could explain the discrepancy. Also, remnants of trees killed by rust early in life in this heavily infected stand may not have been detected.

Sampling among volunteers in the Priest River planting is suspect because silvicultural thinning was carried out in the naturally seeded stand, and this may have biased the results.

Only at Deception Creek were substantial numbers of trees sampled over the range of ages encountered. Here the infection rate increased with tree age. This may be due to the increase in susceptible tissue area as trees grow larger (McDonald and others 1981). Also, the amount of natural inoculum in an area varies from year to year, and wave years of heavy infection occur at irregular intervals when environmental conditions are favorable for development, distribution, and germination of sporidia. In this case, it may be that trees under 15 years old have not been exposed sufficiently to wave infection years. The oldest age group (21 to 25 years), the same ages as the planted pines, matched the infection rate (0.089) of the planted controls.

In any event, these data suggest that if natural white pine regeneration is to be used to assess blister rust hazard of an area:

1. Sampling should be careful and thorough. Particular attention should be paid to locating dead white pines and examining the base of the stems for possible cankers.
2. Sampling should be concentrated on the 16- to 20-year age class if such trees are available. Younger trees may not have been sufficiently exposed to heavy infection years. For older regeneration, it may not be possible to get a satisfactory count of early rust mortality.
3. If hazard at two or more locations is to be compared, the comparison should be based on trees of similar age.
4. Attention should be focused on site factors controlling the degree that infection will deviate from 100 percent in susceptible populations.
5. Concentration should be on infection rate because rust-caused mortality appears to be an unreliable indication of rust hazard. Not only is early mortality difficult to accurately assess in stands over 20 years old, but the amount of mortality appears to be strongly influenced by the age at which infection first occurs.

SUMMARY AND RECOMMENDATIONS

None of the locations in this study can be classed as having low rust hazard. Average infection rates of 11- to 20-year natural regeneration were: Deception Creek—0.06; Priest River—0.09; and Emerald Creek—0.35. If all classes and ages of material are included, one must conclude that Priest River and Deception Creek are about equal, and Emerald Creek is a very high-hazard location. Even a 0.05 annual infection rate translates to 63 percent infection by age 20 and over 90 percent infection and perhaps 50 percent mortality by age 50. Thus, satisfactory yields at rotation should not be expected with use of nonresistant stock at any of the three sites or sites like them.

Open-pollinated progenies of trees selected for resistance but not good general combiners had little superiority over the control lots. Also, the average resistance of open-pollinated progenies of all select trees combined, even though some of them were good combiners, was not high enough to justify their use in areas with infection rates as high as 0.05. Rust-caused losses related to the use of nonresistant stock or open-pollinated seed from untested phenotypically resistant parents would perhaps be tolerable in areas with an expected infection rate less than 2 percent.

Open-pollinated progenies of proven good combiners showed appreciable field resistance in these tests. Seed from such trees should be acceptable for planting sites of low-moderate hazard (sites on which nonresistant trees have 0.03 to 0.06 infection rates).

Crosses among good general combiners such as progenies of seed orchards containing only proven first-generation clones have adequate resistance for planting sites of moderate hazard such as the test sites at Priest River and Deception Creek. Sufficient trees should survive through a rotation of 60 years or more to provide satisfactory yields. However, on sites with hazard as great as that at Emerald Creek, trees with even this level of resistance may not achieve expected yields. Planting of sites showing a greater infection rate than Emerald Creek should definitely be restricted to the products of more advanced rust resistance breeding.

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Three white pine plantations, composed of materials of several levels of resistance from early generations of the blister rust resistance breeding program, were compared on the basis of annual infection and mortality rates. The sites varied in blister rust hazard; resistant materals were consistent in their performance, and the infection data behaved as one would expect if the genetic and environmental assumptions of the simple interest disease progress model were violated.

KEYWORDS: disease resistance, epidemiology, disease-hazard indexing, infection rate, epidemic analysis

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